

### **Listing of Claims**

Following listing of claims is submitted to replace all the prior listings of claims in this application.

1-17 CANCELLED.

18. (CURRENTLY AMENDED) A method for evaluating responsiveness of an individual to treatment with an in vivo pharmaceutical wherein the in vivo pharmaceutical is one which activates G protein heterodimers containing a G protein subunit Gbeta3 or Gbeta3s comprising evaluating the individual for a genetic modification in a gene encoding a Gbeta3 subunit of a protein by detecting the genetic modification in the nucleic acid comprising SEQ ID NO: 2, wherein the genetic modification is a substitution of cytosine by thymidine at position 825 ~~and/or at position 1429~~ of SEQ ID NO:2, and wherein the thymidine at position 825 of SEQ ID NO: 2 is indicative of the individual having increased activation capacity of G proteins which is indicative of an altered responsiveness of the individual to the treatment with the in vivo pharmaceutical as compared to an individual having a cytosine at position 825 of SEQ ID NO:2.
19. (CURRENTLY AMENDED) A method for evaluating responsiveness of an individual to treatment with in vivo hormones, transmitters, neurotransmitters or pharmaceuticals which activate those G protein heterotrimers which contain the G protein subunits Gbeta3 and Gbeta3s and/or which stimulate the G protein subunit GalphaS comprising evaluating the individual for a genetic modification in a gene encoding a Gbeta3 subunit of a protein, wherein the genetic modification is a substitution of cytosine by thymidine at position 825 ~~and/or at position 1429~~ of SEQ ID NO:2, wherein the thymidine at position

825 of SEQ ID NO: 2 is indicative of altered responsiveness of the individual to the treatment with the in vivo hormones, transmitters, neurotransmitters or pharmaceuticals which activate those G protein heterotrimers which contain the G protein subunits Gbeta3 and Gbeta3s and/or which stimulate the G protein subunit GalphaS as compared to an individual having a cytosine at position 825 of SEQ ID NO:2.

20. (PREVIOUSLY PRESENTED) The method of claim 18 or 19, further comprising determining the presence of the Arg16Gly variant and the Gln27Glu variant in the beta2 adrenergic receptor.
21. (CURRENTLY AMENDED) The method of claim 18, wherein the pharmaceutical is erythropoietin.
22. (CURRENTLY AMENDED) The method of claim 18, wherein the pharmaceutical is an immunosuppressive and the development of hypertension during ~~such therapy~~ said treatment is evaluated.
23. (PREVIOUSLY PRESENTED) The method of claim 22, wherein the immunosuppressive is cyclosporin.
24. (PREVIOUSLY PRESENTED) The method of claims 19 or 20, wherein the pharmaceutical is for treatment and prevention of a migraine headache.
25. (CURRENTLY AMENDED) A method for evaluating responsiveness of an individual to treatment with beta-adrenoceptor blockers comprising evaluating the individual for a genetic modification in a gene encoding a Gbeta3 subunit of a human G protein, wherein the genetic modification is a substitution of cytosine by thymidine position 825 ~~and/or position 1429~~ of SEQ ID NO:2, wherein the presence of thymidine at position 825 of

SEQ ID NO: 2 is indicative of the individual having intensified reduction of the cardiac output as a response to treatment with beta-adrenoceptor blockers.

26. (CURRENTLY AMENDED) A method for evaluating responsiveness of an individual in treatment with a substance having prostoglandin E1 action comprising evaluating the individual for a genetic modification in a gene enclosing a Gbeta3 subunit of a human G protein, wherein the genetic modification is a substitution of cytosine by thymidine position 825 ~~and/or position 1429~~ of SEQ ID NO:2, wherein the presence of thymidine at position 825 of SEQ ID NO: 2 is indicative of the individual being less responsive to the substance having prostaglandin E1 action.

27. (PREVIOUSLY PRESENTED) The method of claim 26, wherein the substance is prostaglandin E1.

Claims 28-40 CANCELLED.